DISSERTATION ON

MATERNAL PERIODONTITIS AND ADVERSE PREGNANCY OUTCOMES

Submitted to

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CERTIFICATE

This in to certify that the dissertation work titled **"MATERNAL PERIODONTITIS AND ADVERVSE PREGNANCY OUTCOMES"** is a bonafide research work of DR**. M.SUBHASINI,** Enrolment No…….. Submitted in partial fulfillment of the requirements for the award of Degree of **M.D. OBSTETRICS & GYNAECOLOGY** in **THE TAMIL NADU DR.M.G.R. MEDICAL UNIVERSITY** CHENNAI- 600 032.

Signature of H.O.D **Signature of Dean**

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INTRODUCTION

INTRODUCTION

MATERNAL PERIODONTAL INFECTION AND ADVERSE PREGNANCY OUTCOMES

Despite advances in maternal prenatal care and increased public awareness the Incidence of preterm birth has not decreased over the last 40 years.

This is probably because we have not yet identified all possible causes of preterm births.

As a part of the search, of all identifiable risk factors for Preterm births recently, the role of periodontitis as one of the causation of Preterm births has been extensively evaluated.

Periodontal Infections are relatively common among pregnant women for example occurring at about 2 to 3 times more often than genito - urinary tract Infections.

And the magnitude of risk that is attributable to maternal periodontal disease is as large as other well established risk factors like smoking and lower Genital tract infections.

This Dissertation is in appreciation for the role of distant oral infections and its impact on pregnancy outcomes particularly its influence on birth weight and on gestational age and also to analyse the effect of treatment for clinical periodontitis on the gestational age and birth weight

CLINICAL PERIODONTITIS

Periodontal disease creates a significant systemic inflammatory and microbial assault to the host.

The bacterial load against a ulcerated sulcular epithelium can exceed $10^9 – 10^{10}$ total bacteria in a patient with periodontal disease.

Thus periodontal disease may serve as a potential reservoir for inflammatory cytokines to reach systemic circulation

The chronic and cyclic nature of the periodontal Infection provides opportunity for repeated hematogenous dissemination of periodontal pathogens and hence direct exposure of the vasculature, liver and the PLACENTAL—FETAL UNIT among pregnant women.

THE FETAL INFLAMMATORY RESPONSE SYNDROME (FIRS)

- This term was coined to define a subclinical condition originally described in fetuses of women presenting with PTL and PPROM

- Fetal Microbial invasion or other insults result in systemic inflammatory response that can progress towards multiple organ dysfunction (MOD), septic shock and perhaps death in the absence of timely delivery

AIM OF THE STUDY

AIMS AND OBJECTIVES

Aims

1) To find out the prevalence of periodontits in Pregnant women and to find out the effect of treatment in these women with periodontal infection.

2) Prevalence of periodontits in post natal women who had delivered preterm / LBW, I UGR babies.

Group I –Study of periodontitis in AN Mothers:

Inclusion criteria

All pregnant woman attending AN clinic between GA of 20-28 wks.

Exclusion criteria

1) AN Women < 20 weeks
   Or
   >28 wks gestation
2) Patients with medical disorders.
3) H/O antibiotic therapy during pregnancy
4) Multiple gestation
5) Patients with other obstetric complications.

Group-II

Study of periodontitis in postnatal mothers with LBW / PT babies:

INCLUSION CRITERIA

1) PN mothers who had delivered LBW babies within one day postpartum (ie) <2500gm

2) PN mothers with PT babies – GA <37wks.

EXCLUSION CRITERIA

1) PN mothers with Term babies >37wks and B.wt >2.5kg

2) PN mothers with other medical disorders.

MATERIALS & METHODS

METHODOLOGY

1) All AN women between the Gestation of 20-28 wks attending the AN OP of KMCH are screened for periodontal infection.

2) Periodontal examination was be done by the qualified dentist of dental dept. KMCH.

3) Probing depth is the main criteria for assessing evidence of periodontitis.

4) Those patients with probing depth of >3 mm will be taken as positive cases for periodontitis (involving at least 2 sites)

5) Women positive for periodontitis are given

    1. Oral prophylaxis with Metronidazole 600 mg for 3 days.
    2. Scaling done

6) All pregnant women found positive and treated and along with the normal oral hygiene are followed up till term

7) Pregnancy outcome measured in terms of
    1. Preterm Births GA < 37 wks L.B.W
    2. LBW – Birth weight < 2500 gms
    3. Features of IUGR

8) All post – natal patients with LBW, preterm, IUCR basics are screened for periodontitis similarly.

REVIEW OF LITERATURE

REVIEW OF LITERATURE

OCAP – ORAL CONDITIONS AND PREGNANCY

- A 5 year prospecitive study of pregnant woman designed to determinte the relationship showed a prevalence in mild disease 3.5% and severe disease 11%

- Mitchell Lewis et al in 2001 published results from a group of low socio economies status patients from Newyork. With periodontal infection who received treatment. The prevalence of preterm and LBW was low in the group which received oral prophylaxis

- Jeffcoat and colligues (2003) have proved through studies conducted in antenatal woman by assigning them to one of protocols

  1. Simple teeth cleaning
  2. Antimicrobial therapy with Metronidazole with scaling (or)
  3. Deep root scaling and planing

The Incidence of preterm births was significantly low in the treated group

- Goepfert and colleagues (2004) observed that women who delivered spontaneous before 32 with were significantly more likely to have severe periodontal disease than others.

- Boggess an co- workess (2001) found that preterm NBs of mothers s with periodontal disesase were 23% smaller than those of mothers without disease.

- Finally, 2 multicentre trials sponsored by national Institutesssss of dental and cranio- facial research are underway to determine whether midpregnoncy periodontitis treatment prevents preterm birth.

THE ROLE OF INFECTION AND INFLAMMATION IN PRETERM LABOUR AND BIRTH

Although the multifactorial etiology of preterm delivery is well recognisized apart from the well recognized urinary tract, genital tract Infections ORAL CAVITY is a newly recognised potential source of distant infection for the feto placental unit.

The term parturition describes the changes that occur in preparation for, during and after labor.

In contrast, labor is an active process lasting only hours, by which the fetus is expelled from the uterus.

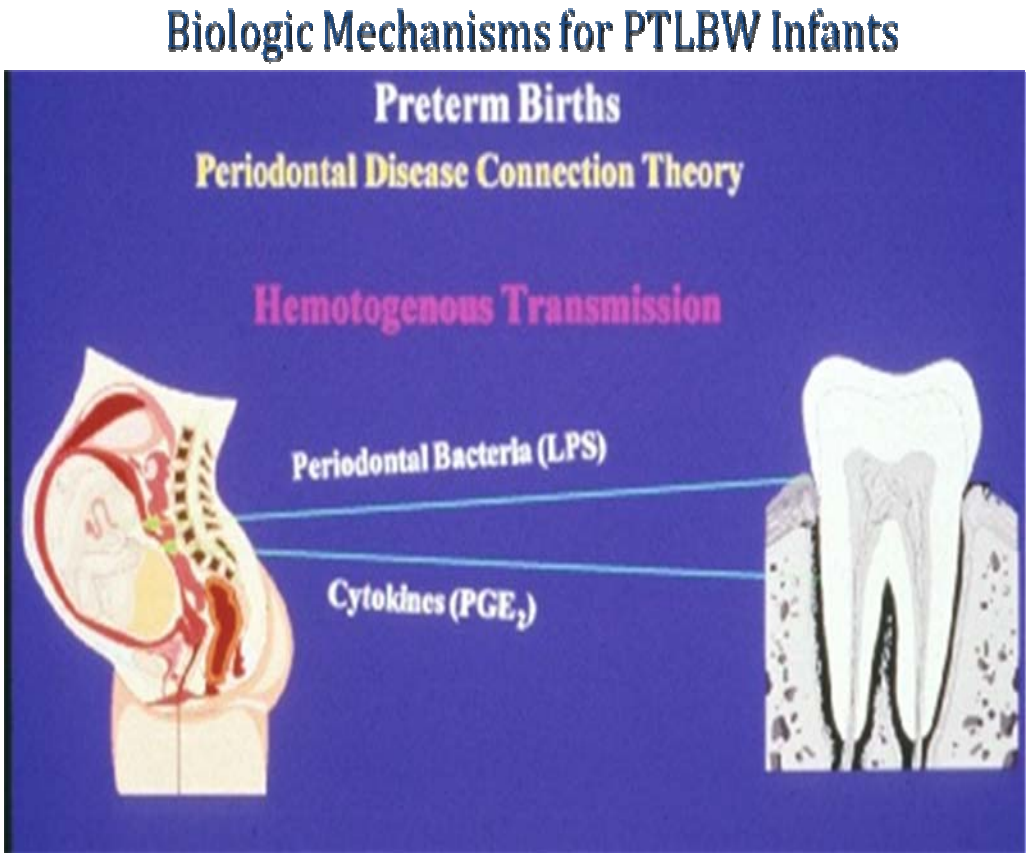
BOTH NORMAL AND PRETERM PARTURITION HAVE 3 INTRAUTERINE PHASES

a. Increased uterine contractions

b. Cervical ripening

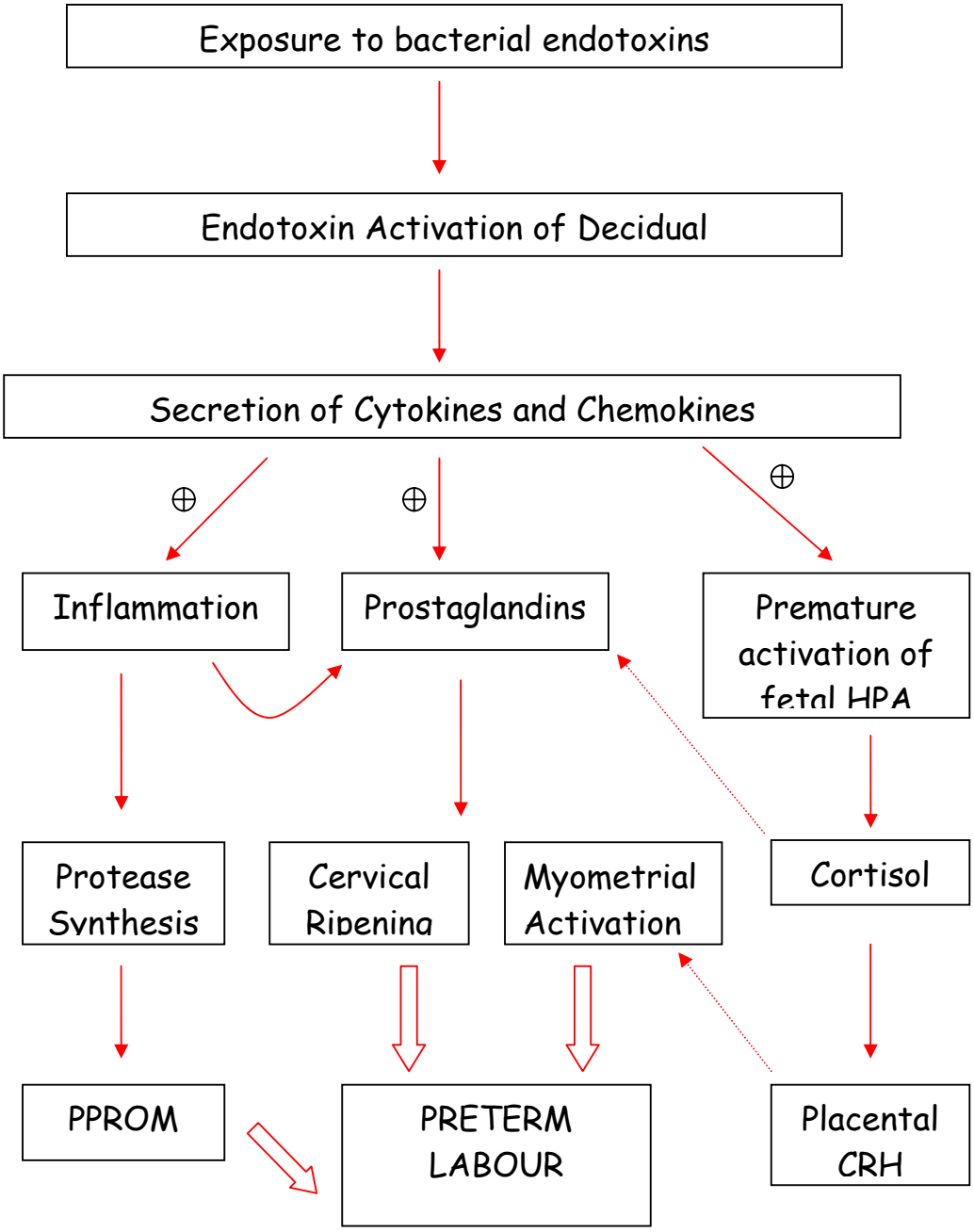
c. Decidual / fetal membrane activation

Inflammatory Mediators Play a Role in The Last 2 Phases



Entry of inflammatory products (PgE2, Il-6, TNF-α), endotoxin, and or periodontal bacteria into the bloodstream and their translocation to the fetus and decidual tissues

INFECTION - INDUCED PRETERM LABOUR



Inflammation of chorioamniotic membranes can result in:

1. Matrix metalloproteinase secretion
2. Mechanical premature rupture

Placental damage- from areas of - inflammation
Results in impaired fetal perfusion and impaired fetal growth

Furthermore,

Increasing evidenced suggests that the molecular and cellular inflammatory effectors pathways that underlet the pathogenesis of preterm birth are also involved in growth restriction and developmental problems

Growth impairment that occurs during gestation can result in a neonate that is small for gestational age (SGA) birth weights $<10^{th}$ percentile of weight for that gestational age

And that gestational age and low birth weighs (ie) < 2.5 kg (for Indian standards)

It has been demonstrated in humans that the periodontal pathogens within the dental plaque are capable of invading into host tissues, eliciting recurrent bacteramias hematogenously translocating to distant tissues and activating the hepatic acute phase response

IMMUNOGY OF ORAL INFECTIONS

Studies have shown that oral organisms were isolated in amniotic fluid, and placental surface

Once at the placental site, P. Gingivalis has been shown to modulate the local Th1/Th2 immune response to impair fetal growth.

A successful pregnancy needs a dominant T_H^2 type immunity with predominated of interleukin-4, IL-10 over TNF- alpha & IFN –gamma

Evidence from pregnant murine infection models demonstrate infection with P.Gingivalis is associated with increase in TNF α and suppression of IL – 10

NET EFFECT

Increase Th1 / Th2 Ratio consistent with Impaired Fetal growth studies have shown an elevated levels of Igm ab's to oral pathogens in fetal cord blood samples.

Aminiotic fluid IL – 6 concentration is a marker of Intra – amniotic inflammation and is frequently associated with microbiologic infection in either amniotic fluid or chorioamniotic space.

- ❖ Pregnancy increases risk for gingivitis and periodontitis, and can accelerate attachment loss, through local increases in:

  - PMNs in the sulcus increasing Vascular Permeability
  - Bacteroides species increasing PgE2
  - Kaulkwarf 78, Kornman 80, Lieff 2004

WHAT IS PERIODONTAL DISEASE?

Periodontal disease consists of both gingivitis and periodontits.

Prevalence varies between 10-60% of adults depending upon the diagnostic criteria.

GINGIVITIS

Defined as an inflammatory condition of the soft tissue surrounding the tooth or the gingiva.

PERIODONTITIS

Destruction of supporting structures such as periodontal ligament attachment is usually associated with loss of supporting alveolar bone with ensuing mobility and potential for loss of teeth.

The disease is initiated by the presence of gram negative bacteria in dental plaque.

PLAQUE

Consists principally of bacteria that are adherent of the teeth and appears as a thin, white translucent slimy film Just above and below the gum line.

CALCULUS

A mature plaque, with non viable dental organisms which becomes calcified.

Oral Disease and Systemic Disorders
Periodontitis and pregnancy

PREGNANCY TUMOUR

BACTERIOLOGY OF PERIODONTITIS

The organisms associated with periodontitis include “RED CLUSTER” and “ORANGE CLUSTER” organisms

ORANGE CLUSTER:

Campylobacter rectus
Fusobacterium nucleates
Prevotella.

RED CLUSTER:

Porphyromonas Gingivalis
Treponema Denticola
Tannerealla Forsythensis

Orange cluster organisms colonise first within the sublingual plaque and provide necessary environment for the more tissue invasive “Red” cluster microbes.

These organisms cohabitate within a glycocalyx rich biofilm that is sensitive to systemic antibiotics.

Thus the systemic administration of antibiotics as monotherapy such a metronidazole would have a significant impact on the microbial component of the dental bio film.

ADVERSE OUTCOMES ASSOCIATED WITH PERIODONTITIS

1. Preterm births
2. Intrauterine growth restriction
3. Preeclampsia
4. SGA
5. Low birth weight < 2500 gm.
6. Chorioamnionitis

Pre-Term, Low Birth Weight Infants:

- < 37 weeks, < 5lbs 8oz
- Related to 60% of infant deaths
- 25% can't be explained by accepted risk factors such as smoking, alcohol, nutrition, UTI, and level of education or prenatal care.

Periodonitis is a newly discovered, potential source of infection to the fetoplacental unit

Oral Disease and Systemic Disorders

PTLBW infant treatment in hospitals for respiratory distress, jaundice, malnutrition, anemia, CP, etc. costs over $5 billion annually.

CLINICAL FEATURES OF PERIODONTITIS

- Halitosis
- Pronounced ease of bleeding even with mild stimulus (brushing)
- Inflammed and reddish looking gums.
- Inflammed gingiva may form discrete tumor like masses called pregnancy tumors
- Loosening of teeth in severe cases

DIAGNOSIS

Periodontitis is diagnosed by performing a professional periodontal examination that involves inserting a periodontal probe between the tooth and gum at 6 sites involving the faucial and lingual surfaces of the teeth.

Depth of periodontal pocket(distance form the gingival margin to depth of periodontal pocket.)

In health - Probing depth 1-3 mm
- With intact epithelial attachment to the tooth
- Periodontal pocket do not bleed on probing

Probing >3mm - Evidence of periodontal disease

OTHER INDICES USED FOR THE STUDY

Simplified - oral hygiene Index (OHI-S)

OHI-S =calculus Index +Debris Index

OHI-S = CI+DI

TREATING PERIODONTITIS

It is Hence forth, worthwhile treating periodontal disease to give a better pregnancy outcome.

WHO 1977.endorsed Treatment Protocol

Periodontal slates	Treatment Needs
O = Health Periodontium	= No Treatment Needs
1 = Bleeding observed, directly Or using mouth mirror.	1 = oral Hygiene instructions professional
2 = calculus felt during probing	11 = 1 + scaling
3 = Pocket > 3	11 = 1 + Professional scaling + antibiosis
4 + Pocket > 6mm	111 =1+ 11+ surgical treatment

1. In this study patients positive for periodontitis were treated with oral METRONIDAZOLE and under went scaling.

Biologic Mechanisms

- Infection in the chorioamnion may be a cause of pre-term birth. NEJM, Hillier 1988.

- Infection with P. gingivalis increased PgE2 and TNF-α and appeared to be asso. with decreased fetal birth weight in the hamster. Collins, Infec Immun 1994.

- Pg E2 levels in gingival crevicular fluid was sig higher in mothers of LBW infants than in controls. The lower the birth weight, the higher the PgE2. Offenbacher 1998.

Clinical Studies

Periodontal Infection as a Possible Risk Factor for Preterm Low Birth Weight. n=93 PTLBW, 31 normal.

The risk for preterm labor requiring medical intervention or premature rupture of membranes (<36 weeks), or low birth wt. Infants (<2500 grams), was greater if the mother had periodontitis:

- PTB- OR 7.9
- LBW- OR 7.5

(after controlling for smoking, race, alcohol use, age, nutrition, and genitourinary infection.) - offenbacher 1996.

Biologic Mechanism- Immune Response

Maternal periodontal infection in the absence of a protective maternal antibody response is asso with systemic dissemination of oral organisms that translocate to the fetus resulting in prematurity. Highest rate of prematurity of 67%:

- Maternal periodontal infection
- Decreased IgG response to some periopathogens
- Strong IgM fetal response to other periopathogens

(Fetal cord serum samples)

Madianos Annals of Perio 2001

Maternal periodontitis is an independent risk factor for PTB, LBW, and fetal growth restriction.

5 Year prospective study. n= 814

Perio exams <26 weeks and w/i 48 hrs postpartum.

Prevalence of births for gestational age <28 weeks:

- Perio healthy - 1.1%
- Mild periodontitis- 3.5%
- Mod-severe perio- 11.1%

Prevalence of LBW deliveries at < 1000 grams

- Perio healthy- 0%
- Mild periodontitis- 6.1%
- Mod-severe perio- 11.4%

Periodontitis incidence/progression during pregnancy was asso with sig smaller births for gestational age

- Offenbacher 2001

INTERVENTION STUDIES-PTLBW INFANTS

In pregnant mothers with periodontitis, periodontal therapy reduces the risk of PTLBW infants:

- Control (no perio therapy)- Incidence 10.11%
- Therapy before 28 weeks gestation- Incidence 1.84%.

Periodontitis was the strongest risk factor for PTLBW (OR 4.70), compared to previous PTLBWI (OR 3.98).

Periodontitis is an independent risk factor for PTLBWI, and perio therapy reduces the rates of PTLBW.

-- Lopez 2002, n=351

Periodontitis therapy at 21-25 weeks gestation reduced the rate of spontaneous PTB at < 35 weeks:

No periodontitis rate 6.3%

Periodontitis therapy

Prophy rate 4.9%

SRP + Met rate 3.3%

SRP rate 0.8%

- Jeffcoat 2003

n=366, w 723 controls

Conclusions- Periodontitis and PTLBW Infants

- Periodontitis appears to be an independent risk factor for pre-term delivery.

- There is a clear need for more well designed observational and intervention studies to confirm observations.

Expectant mothers should be counselled about the importance of oral health.

Dental Considerations

- Preventive oral care services should be provided as early in pregnancy as possible.

- If exam indicates a need for periodontal therapy, these procedures should be scheduled early in the 2nd trimester.

- The presence of acute infection, abscess, or other potentially disseminating sources of sepsis may warrant prompt intervention, irrespective of the stage of pregnancy.

Recommendations

- Regular dental examinations for all pregnant patients
- Aggressive periodontal therapy for infections
- Frequent reinforcement of oral hygiene and dental care by medical providers

Review

RESULTS & ANALYSIS

GROUP I - PREVALENCE AND EFFECT OF TREATMENT OF PERIODONTITIS IN ANTENATAL WOMEN

TABLE: 1

PERIODONTITIS	NO	%age
PRESENT	16	10.7%
ABSENT	134	89.3%
TOTAL	150	100%

TOTAL STUDY GROUP – 150

TOTAL CASES POSITIVE FOR PERIODONTITIS - 16

PREVALENCE OF PERIODONTITIS IN ANTENATAL WOMAN WAS - 10.7%

AGE DISTRIBUTION OF PERIODONTITIS

TABLE: 2

AGE GROUP	EVIDENCE OF PERIODONTITIS		NO EVIDENCE		TOTAL STUDY GROUP	
	NO.	% age	No	% age	No	% age
15-20 yrs	2	1.3%	20	13.3%	22	14.7%
21-25 yrs	11	7.3%	89	59.3%	100	66.7%
26-30 yrs	2	1.3%	22	14.7%	24	16.0%
30-35 yrs	1	0.7%	3	2.0%	4	2.7%
TOTAL	**16**	**10.7%**	**134**	**89.3%**	**150**	**100%**

10.7% OF THE STUDY GROUP HAD PERIODONTITIS & AMONG THEM 7.3% WERE 21-25 YEARS OLD & 0.7% WERE >30 YEARS

PEARSON CHI- SQUARE TEST – P VALUE 0.271

NO STATISTICALLY SIGNIFICANT DIFFERENCE WITH REGARD TO AGE

RESIDENCE DISTRIBUTION OF PERIODONTITIS

TABLE: 3

RESIDENCE	EVIDENCE		NO EVIDENCE		TOTAL	
	NO	% age	NO	% age	NO	% age
SEMI URBAN	10	6.7%	99	66%	109	72.7%
RURAL	4	2.7%	24	16%	28	18.7%
URBAN SLUM	1	0.7%	10	6.7%	11	7.3%
URBAN DEVELOPED	1	0.7%	1	0.7%	2	1.4%

AMONG THE 16 CASES (10.7) PERIODONTITIS 6.7% BELONGED TO SEMI URBAN POPULATION.

PEARSON CHI SQUARE TEST – P VALUE - 0.551

NO STATISTICALLY SIGNIFICANT DIFFERENCE WITH REGARD TO RESIDENCE.

RELIGION DISTRIBUTION OF PERIODONTITIS

TABLE: 4

RELIGION	EVIDENCE		NO EVIDENCE		TOTAL	
	No	% age	No	%age	No	%age
HINDU	13	8.4%	74	51.7%	87	59.1%
CHRISTIAN	2	1.6%	30	20.11%	32	21.7%
MUSLIM	1	0.8%	25	17.0%	26	17.8%
OTHERS	0	0%	2	1.4%	5	1.4%

OUT OF THE STUDY GROUP OF 150, 59.1% WERE HINDUS.

10.7% of the study group had periodontitis among them 8.4% were hindus
P VALUE 0.551

NO STATISTICAL DIFFERENCE WITH REGARD TO RELIGION

PARITY DISTRIBUTION

TABLE: 5

PARITY	EVIDENCE		NO EVIDENCE		TOTAL	
	No	% age	No	% age	No	% age
PRIMI 'S	15	8.1%	114	49.7%	129	86.6%
G2	1	0.7%	15	10.1%	16	10.7%
G3	0	0%	1	0.7%	1	0.7%
G4 & ABOVE	0	0%	1	0.7%	1	0.7%

IN THE STUDY GROUP OF 150 – 86.6% WERE PRIMIS

10.7% OF THE STUDY GROUP HAD PERIODONTITIS 8.1% OF THEM WERE PRIMIS.

WHEN COMPARED TO OTHERS THERE WAS NO STATICAL SIGNIFANCE WITH REGARD TO PARITY.

SYMPTOMS OF PERIODNTITIS – DISTRIBUTION

TABLE: 6

SYMPTOMS	EVIDENCE		NO EVIDENCE		TOTAL	
	No	**% age**	**No**	**% age**	**No**	**% age**
SYMPTOMATIC	7	4.7%	21	14%	28	18.95%
ASYMPTOMATIC	9	6%	113	75.3%	122	81.3%

OUT OF THE 10.7% CASES OF PERIODONTITIS 6% WERE ASYMPTOMATIC.

P VALUE 0.086

STATISTICAL SIGNIFANCE IS PRESENT

HENCE ASYMPTOMATIC WOMEN ALSO HAD PERIODONTITIS

BIRTH WEIGHT DISTRIBUTION

TABLE: 7

BIRTH WEIGHT	PERIODONTITIS TREATED GROUP		NO PERIODONTITIS		TOTAL	
	No	% age	No	% age	No	% age
1100-1.5	1	0.7%	1	0.7%	2	1.4%
1.6-2.0	1	0.7%	6	4.0%	7	4.7%
2.1-2.5	4	2.7%	28	18.7%	32	21.3%
2.6 – 3.0	10	6.7%	87	58.0%	97	64.7%
3.1 – 3.5	0	0%	11	7.3%	11	7.3%
>3.5 kg	0	0%	1	0.7%	1	0.7%

T-TEST

PERIODONTITIS	N	MEAN BIRTHWEIGHT	S.D	S.E
TREATED	16	2.5219	0.43165	0.10791
NO EVIDENCE	134	2.7194	0.37129	0.03207

MEAN BIRTH WEIGHT AMONG PERIODONTITIS TREATED GROUP - 2.5 kg

MEAN B.wt IN THE NON PERIODONTITIS GP – 2.7 KG
P VALUE - 0.05

NO STATISTICALLY SIGNIFICANT DIFFERENCE BETWEEN THE 2 GROUPS

GESTATIONAL AGE DISTRIBUTION

TABLE: 8

GA	PERIOD TREATED GROUP		NO PERIODNTITIS		TOTAL	
	No	% age	No	% age	No	% age
>37 wks	14	9.3%	118	78.7%	132	88%
34-36 wks	0	0%	13	8.7%	13	8.7%
30-33 wks	2	1.4%	3	2.0%	5	3.3%

T-TEST

PERIODONTITIS	N	MEAN GA	S.D	S.E
TREATED	16	>37 wk	0.68313	0.17078
NO EVIDENCE	134	>37 wk	0.40953	0.03538

P- VALUE – 0.359

GA WAS NOT STATISTICALLY SIGNIFICANT BETWEEN

PERIODNTITIS TREATED GROUP AND NO EVIDENCE GROUP

GROUP: II PERIODONTAL SCREENING OF POSTNATAL WOMEN WITH LBW / PT BABIES

PERIODONTITIS	NO	% AGE
PRESENT	6	12.0%
ABSENT	44	88.0%
TOTAL	**50**	**100%**

PREVALENCE OF PERIODONTITIS IS 12%

AGE DISTRIBUTION

TABLE: 2

AGE	EVIDENCE		NO EVIDENCE		TOTAL	
	NO	% age	NO	% age	NO	% age
15-20 yrs	2	4.0%	4	8%	6	12%
21-25	1	2.0%	29	58%	30	60%
26-30	0	0%	10	20%	10	20%
31-35	3	6%	1	2%	4	8%

12% OF THE STUDY GROUP HAD PERIODONTITIS, AMONG THEM 4% BELONGED TO AGE GROUP OF 15 TO 20 YEARS.

ON THE OTHER HAND, 6% OF THE STUDY GROUP WITH PERIODONTITIS BELONGED TO AGE GROUP OF 31 TO 35 YEARS

NO STATISTICAL SIGNIFANCE DIFFERENCE WAS NOTED

RESIDENCE DISTRIBUTION

TABLE: 3

RESIDENCE	EVIDENCE		NO EVIDENCE		TOTAL	
	NO	% AGE	NO	% AGE	NO	% AGE
SEMI URBAN	4	8.0%	18	26%	22	32%
RURAL	1	2.0%	16	28.5%	17	30.5%
URBAN SLUM	1	2.0%	13	21.5	14	23.5%
URBAN DEVELOPED	0	0%	7	14.0%	7%	14.0%

12% OF THE STUDY GROUP HAD PERIODONTITIS, 8% BELONGED TO SEMI URBAN.

THE PREVALNCE OF PERIODONTITIS WAS MORE IN THIS POPULATION

RELIGION DISTRIBUTION

TABLE: 4

RESIDENCE	EVIDENCE		NO EVIDENCE		TOTAL	
	NO	% age	NO	% age	NO	% age
HINDUS	6	12%	39	78%	45	90%
CHRISTIANS	0	0%	5	10%	5	10%

ALL PATIENTS IN THE PERIODONTITIS GROUP WERE HINDUS HENCE STATISTICALLY SIGNIFICANT DIFFERENCE EXISTS.

PARITY DISTRIBUTION

TABLE: 6

PARITY	EVIDENCE OF PERIODONTITIS		NO EVIDENCE		TOTAL	
	NO	% age	NO	% age	NO	% age
PRIMI	2	4.0%	34	68%	36	72%
G2	1	2.0%	5	10.0%	6	12%
G3	2	4.0%	5	10.0%	7	14%
G4 2 ABOVE	1	2.0%	0	0%	1	2%

4% OF THE STUDY GROUP WHO HAD PERIODONTITIS WERE PRIMI

NO STATISTICALLY SIGNIFICANT DIFFERENCE WAS OBSERVED WITH REGARD TO PARITY.

BIRTH WEIGHT DISTRIBUTION

TABLE: 7

BIRTH WEIGHT	PERIODONTITS GROUP		NO EVIDENCE		TOTAL	
	NO	% age	NO	% age	NO	% age
1-1.5 kg	0	0%	2	4%	2	4%
1.6-2.0 kg	2	4%	1	2%	3	8%
2.1 – 2.5 kg	3	6%	5	2%	8	4%
2.6 – 3.0 kg	1	2%	31	66%	32	72%
3.1 – 3.5 kg	0	0%	5	2%	5	4%

IN THE PERIODONTITIS GROUP – 5% BABIES WERE LOW BIRTH WEIGHT (i.e) < 2.5 kg

T – TEST

PERIODONTITIS	N	MEAN BIRTH WEIGHT	S.D	S.E
PRESENT	6	2.3 kg	0.258	0.10567
ABSENT	44	2.8kg	3.136	0.0472

P VALUE => 0.005

THERE WAS STATICALLY SIGNIFICANT DIFFERENCE IN B.WT BETWEEN PERIODONTITIS & NON-PERIODONTITIS GROUP.

GESTATIONAL AGE DISTRIBUTION

TABLE: 8

GA	PERIODONTITIS EVIDENCE		NO EVIDENCE		TOTAL	
	NO	% age	NO	% age	NO	%age
>37 WKS	2	4.0%	35	72%	37	76%
34-36	2	4.0%	5	10.0%	7	14%
30-33	2	4.0%	3	6%	5	10%
< 30 wks	0	0%	1	2%	1	2%

* PREVALENCE OF PRETERM IN PERIODONTITIS GROUP – 8%

T- TEST

PERIODONTITIS	N	MEAN GA	S.D	S.E
PRESENT	6	36wks	2.892	1.1832
ABSENT	44	37wks	2.525	0.3806

P VALUE => 0.228

NO STATISTICAL SIGNIFICANT BETWEEN TWO GROUPS REGARDING GA.

COMPARISON OF THE PREVALENCE OF PERIODONTITIS BETWEEN AN & PN GROUP

TABLE: 9

PERIODONTITIS	AN		PN	
	NO	**% AGE**	**NO**	**% AGE**
PRESENT	16	10.7%	6	12%
ABSENT	134	89.3%	44	88%
TOTAL	**150**	**100%**	**50**	**100%**

P VALUE – 0.005

NO DIFFERENCE IN PREVALENCE OF PERIODONTITIS BETWEEN AN & PN GROUPS

DISCUSSION

The study of prevalence of Periodontitis in pregnant women was started in the AN OP of K.M.C involving women Between GA of 20-28 wks OTHER infections as a cause of LBW / PT births were RULED OUT.

GROUP I - Periodontitis in AN mothers

- Out of 150 women studied
- 16 cases were found positive for periodontitis
- Prevalence was 10.7%
- Regarding Age Distribution
- 66.7% of the study group Belonged to 21 -25 yrs.

❖ 72.7% of the study population belonged to the semiurban localily There was No significant different Regarding .Age , Resdience, and parity in the Occurance of periodontitis.

❖ 59.1% of the study group were hindus, of them 8.4% had periodontitis (72.7%) hence Religion has no significance.

❖ In the study group of 150, 81.3% were Asymptomatic of them 6% had Evidence of periodontitis.

❖ Hence majority of the patients with periodontitis were Asymtomatic Hence the need to screen all AN women even those without symptoms.

- All women with periodontitis were treated hence as expected the prevalence of low birth weight among the periodontitis Treate group was only 1.4% which was grossly low.

- The mean birth weight among the periodontitis treated group was 2.52 kg. mean GA – 37 wks.

- There was no statistically significant difference between the periodontitis positive and negative groups.

GROUP - II - Periodontitis in PN mothers

- The prevalence of periodontitis among those postnatal women who delivered either LBW or preterm birth was 12%.
- Like the AN group, in age, Residence, Religion and Occupation there was no statistically significant difference between the periodontitis Positive and negative group.
- Out of the total 50 cases studied 6 were positive for periodontitis 72% of the study group were primis 4% of the primis suffered from periodontitis 2% of the G2's and 4% of the third Gravida suffered from periodontitis.
- Out of 6 cases positive for periodontitis -5 pts had delivered LBW or PT babies.
- They had no prenatal screening or treatment or were they symptomatic.
- Mean birth weight among the periodontitis group 2.3kg and B.lot among the Non periodontitis group was 2.8 kg. There were statistically significant difference between the 2 groups.
- The prevalence of periodontitis was comparable between AN and PN groups.
- As with other studies, this study has also proved that prevalence of PT. LBW was low among the group which was treated for the infection.

This stresses the need for oral hygiene during pregnancy and routine screening for periodontal ds. To reduce the incidence of adverse outcomes like PTB.

SUMMARY

GROUP I

- Prevalence of periodontitis in AN women = 10.7%
- Incidence of LBW infants in the
  a) AN periodontitis treated group -4.1%
  b) Non periodontitis group – 23.4%

- incidence of PT infants
  a) AN periodontitis treated group -1.4%
  b) Non periodontitis group – 10.7%

- Mean B.wt among the perodontitis treated gp =2.5 kg
- Mean GA in the same gp >37 wks

GROUP II

- Prevalence of periodontitis in PN mothers with LBW/ IUGR Babies =12%.

- Mean B.wt among the perodontitis -2.3kg.

- Mean GA in the same gp - 36 wks.

- No statistically significant difference with regard to age residenc, Religion and parity.

NO STATISTICALLY SIGNIFICANT DIFFERENCE IN GA/B.WT BETWEEN AN PERODONTITIS TREATED AND NO EVIDENCE GROUP.

CONCLUSION

Among the various infection implicated in the cause of PTB/ LBW- periodontitis is a relatively new one.

In this study, prevalence of periodontitis and is comparable between AN and PN groups.

Treating periodontitis, has significantly reduced the incidence of LBW and PT birth.

Mid- Pregnancy identification and treatment of periodontitis in all AN women proves to be effective.

This dissertation thus emphasizes the need to screen all AN woman for periodontitis to reduce adverse pregnancy outcomes.

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PROFORMA

Maternal periodontitis and adverse Pregnancy Outcomes- PROFORMA
Name - Age - Serial No –
Husbands Name
IP.No.
Type of the Subject – case / control.
Address -

In come - Occupation -

Obstetrics Score: G P L A
LMP
EDD –

Present obstetrics History –

H/o Months amenorrlora

Prev. obstetrics History –
H/o Months amenorrlora

Prev. obstetric History - Full Team / Preterm

Noc –
Abortion –
LCB –
Contraception.

Any Dental Check up in the past.

Family History –

Past Medical / Surgical history

Dental History.

H / o oral Prophylaxis
H / o periodontal Surgery

H / o Tooth Extraction

Habits:

Brushing Hand / Brush user tooth Powder / Tooth Paste
Frequency of brushing once / Twice

H /o Bleeding gums
H /o Swollen gums
H /o carious – if so – how many?

Others:

- H /o U T I
- H / o Localized septs

General Examination

Anaemia Ht :
Pedal Oedema Wt :
T
PR
BP
Breast, Thyroid, spine

P/A –

P/V –

Labour:

DENTAL EXAMINATION

Probing Depth - Normal (0-3mm)

Calculus Index -

Debrin Index -

OHI –S - CI + DI

Evidence of Periodontitis +/-

Peritnatal outcome :

DOB -
Sex -

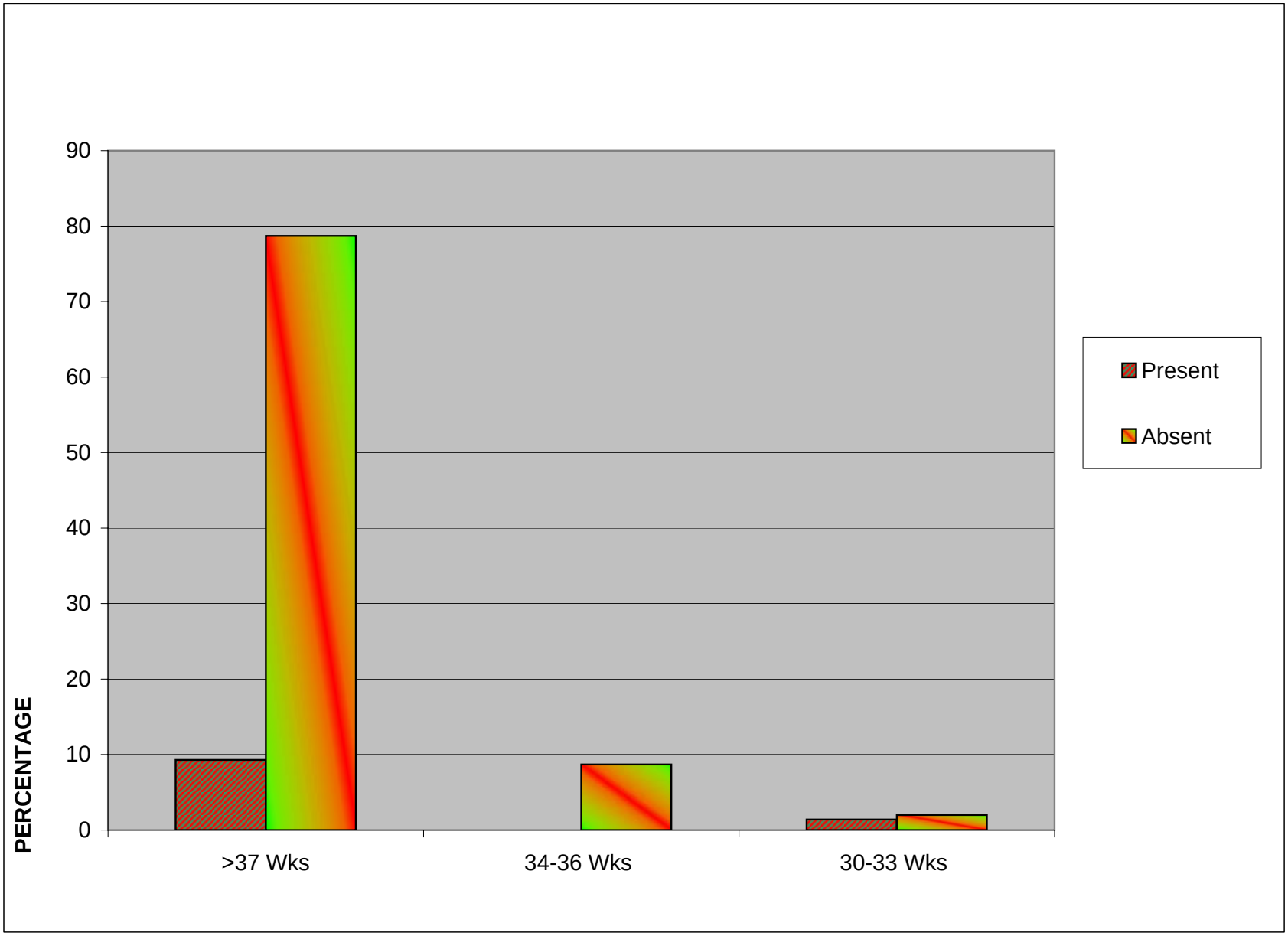
Geotational age -

Apgar –
B.wt -

Admission on is NICU
Complication -

ABBREVIATIONS

AN	-	ANTENATAL
PN	-	POSTNALAL
LBW	-	LOW BIRTH WEIGHT
PTB	-	PRETERM BIRTH
GA	-	GESTATIONAL AGE
PTL	-	PRETERM LABOUR
SRP	-	SCALING ROOT PLAINING
FIRS	-	FETAL INFLAMMATORY RESPONSE SYNDROME


90
80
70
60
50
40
30
20
10
0
PERCENTAGE
>37 Wks
34-36 Wks
30-33 Wks
Present
Absent

	AN	PN
PRESENT	10.7	12
ABSENT	89.3	88

SL.NO	NAME	IP NO	AGE	RESIDENCE	RELIGION	EDUCATION	OCCUPATION	PARITY	BW GROUP	BIRTH WEIGHT	GA GROUP	PD	EVIDENCE
1	Janay Rani	1562	22	2	1	2	6	1	5	2.1	1	0	2
2	Ranuka	1752	24	1	2	2	6	1	4	1.8	3	0	2
3	Parameshwari	1560	26	1	3	2	5	1	5	2.5	2	0	2
4	Sasi	1672	30	1	4	3	6	2	5	2.3	1	1	2
5	Valli	1608	24	1	1	3	6	1	4	1.9	1	1	2
6	Usha	1674	21	1	2	3	6	2	4	1.9	2	0	2
7	Prema	1656	18	1	2	2	6	1	5	2.4	3	0	2
8	Humalatha	1655	21	1	2	2	3	3	5	2.5	2	0	2
9	Thara	1660	18	1	1	3	6	3	5	2.2	1	0	2
10	Jayalakshmi	1678	22	1	2	1	6	1	5	2.4	1	0	2
11	Adhilakshmi	1678	23	1	3	2	5	3	5	2.3	1	0	2
12	Mariamma	1525	24	1	1	2	5	1	4	1.8	1	0	2
13	Soundari	1542	25	1	3	2	4	1	5	2.4	1	0	2
14	Kalaiselvi	1672	32	1	1	2	3	3	4	1.9	1	4	1
14	Devi puroshatai	1672	32	1	1	2	3	3	4	1.9	1	4	1
15	Shakthi	1701	22	1	2	3	2	1	4	1.9	2	4	1
16	Indra	1705	23	1	3	2	3	1	5	2.3	1	0	2
17	Sayatha	1692	22	1	1	3	5	1	5	2.5	1	0	2
18	Sumathy	1678	24	1	2	2	6	2	5	2.4	2	0	2
19	Kanchana	1656	22	1	2	2	3	1	5	2.4	1	0	2
20	Kalyani	1672	21	1	2	2	3	1	5	2.4	1	0	2
21	Ganga	1709	27	1	1	3	5	1	5	2.4	1	0	2
22	Elizabeth	1598	28	1	2	3	6	1	3	1.2	4	0	2
23	Angammal	1600	24	2	1	3	5	3	5	2.2	1	0	2
24	Navanulham	1616	21	1	2	2	2	1	5	2.3	2	0	2
25	Saraswathi	20144	24	1	1	3	6	1	5	2.1	1	0	2
26	Vanamil	9014	22	1	2	4	3	1	5	2.3	1	0	2

27	Ganamani	9025	21	1	1	3	5	1	4	1.8	3	0	2
28	Devi	9185	22	1	2	3	3	1	5	2.4	2	0	2
29	Revathy	9252	20	1	2	3	3	2	4	1.8	3	1	1
30	Nila	9018	23	1	2	2	5	1	5	2.4	1	0	2
31	Malar	9172	19	2	2	4	3	1	5	2.3	1	0	2
32	Louria Mary	8900	26	1	1	5	2	1	5	2.4	1	0	2
33	Pachamani	9128	21	1	1	5	2	1	5	2.2	2	0	2
34	Anbumani	9128	22	1	1	5	2	1	5	2.3	2	0	2
35	Vidhya	9075	24	1	1	2	6	1	5	2.1	1	0	2
36	Shankari	9072	27	1	2	4	3	1	5	2.3	1	0	2
37	Priya	8905	32	2	3	4	4	2	5	2.4	1	0	2
38	Mathura	8156	30	2	3	4	4	3	5	2.1	2	0	2
39	Kala	9052	28	1	3	3	4	1	5	2.3	1	0	2
40	Venda	9275	22	1	3	3	3	1	5	2.4	1	0	2
41	Vani	9529	18	1	2	3	3	1	4	1.4	3	1	1
42	Prema	9955	24	1	3	2	6	1	5	2.1	1	0	2
43	Gayalakshmi	9255	23	1	1	2	6	2	5	2.5	1	0	2
44	Sonurshwari	995	27	1	3	5	4	1	3	1.4	3	0	2
45	Kanimozhi	9156	26	1	3	5	4	1	3	1.3	3	0	2
46	Vidhya	9156	19	1	1	5	6	1	5	2.4	1	0	2
47	Mookambigai	9256	24	1	2	5	4	1	5	2.3	3	0	2
48	Mariamma	9062	34	1	1	2	6	4	5	2.2	2	1	1
49	Prema	9625	23	1	2	3	3	1	5	2.5	1	0	2
50	Louria Mary	9728	23	1	2	3	3	1	5	2.5	1	0	2